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CHENNAI

A CLINICAL STUDY ON DIAGNOSIS, CLINICOPATHOLOGICAL CORRELATION AND MANAGEMENT OF ACUTE CHOLECYSTITIS

at K.A.P.V Government Medical College & A.G.M.Government Hospital, Trichy.

Dissertation submitted for

M.S.General Surgery [Branch-1], March 2010

CERTIFICATE

This is to certify that the dissertation on "**A CLINICAL STUDY ON DIAGNOSIS, CLINICOPATHOLOGICAL CORRELATION AND MANAGEMENT OF ACUTE CHOLECYSTITIS**" presented herein by Dr.G.MOHANDHAS, is an original work done in the department of General Surgery, K.A.P.V Government Medical College and Hospital, Trichirappali in partial fulfillment of regulations of the Tamilnadu Dr.M.G.R.Medical University for the award of M.S.Degree (General Surgery) Branch-I, under my guidance and supervision during the academic period 2007-2010.

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DECLARATION

I, G.Mohandhas, solemnly declare that this dissertation, titled "**A CLINICAL STUDY ON DIAGNOSIS, CLINICOPATHOLOGICAL CORRELATION AND MANAGEMENT OF ACUTE CHOLECYSTITIS**" is a bonafide record of work done by me in the department of General Secretary, K.A.P.V Government Medical College and Hospital, Tiruchirappalli, under the guidance of **Prof.Dr.G.MURALIDHARAN M.S, Prof.Dr.G.RAJAMBIKAI M.S** Professor of Surgery, Unit Chief, K.A.P.V Governement Medical College AGMGH Hospital, Trichy.

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INTRODUCTION

INTRODUCTION

Acute cholecystitis is the most common complication of gallstone disease, and a frequent cause of abdominal emergency, especially among middle aged women and the elderly.

They are mainly classified into two types:

1. Acute calculous cholecystitis
2. Acute acalculous cholecystitis.

ACUTE CALCULOUS CHOLECYSTITIS:

Acute inflammation of the gall bladder is associated with cystic duct obstruction, which commonly occur due to impacted gall stone in calculous cholecystitis. The combination of cystic duct occlusion and altered biliary lipid composition appears to initiate a series of events culminating in local release of inflammatory agents and ultimately resulting in acute cholecystitis.

ACUTE ACALCULOUS CHOLECYSTITIS:

Acute acalculous cholecystitis is a treacherous and potentially lethal disease. It may occur in patients with no known biliary tract disease who are severely compromised by trauma, gastrointestinal dysfunction, prolonged intensive therapy for sepsis, burns or Multi Organ Failure (MOF). They can lead to fatal life threatening complications like empyema, perforation and gangrene of the gall bladder.

The approach, diagnostic evaluation and management of acute cholecystitis has come miles away from the older days of elective cholecystectomy to the present day hurricane management like emergency open cholecystectomy ; minimally invasive cholecystectomy and laparoscopic cholecystectomy using space age tools like laser cautery.

HISTORICAL ASPECTS

Gallstones are known for centuries. It was found in the mummies of Arnan in 21st Egyptian dynasty - 1085-95 BC.

Gallstone colic was described by Hippocrates and fomentation and Herbal medicine were advised by Galen in II century. Charaka called it Panchak Pitta. Sushruta - said bile was digestive fire and named it Panchak agni.

1667 : *Von der wal* evacuated gallstones on opening the abscess of abdominal wall.

1733 : *Jean Louis Petit* advocated direct incision into gallbladder in acute cholecystitis if gall bladder was adherent to abdominal wall for the drainage of Pus.

1844 : *Duncan* reported fatal case of perforated acalculous cholecystitis after patient underwent surgery for femoral hernia.

1873 : *Lawson Tail* presented a series of cholecystectomies of patients.

1924 : *Graham Cole* described oral cholecystogram.

1980 : Concept of ESWL by *Dormier* in W. Germany

1986: *Sauerback* reported fragmentation of gallstones and reported best results with solitary stones.

1983 : *Gocco and Chambers coined* the term mini cholecystectomy thro mini laparotomy.

1987: First laparoscopic cholecystectomy was done **P.Maaret**

REVIEW OF LITERATURE

ANATOMY:

Understanding the anatomy of the gall bladder and the extra hepatic biliary system is essential for the accurate diagnosis and the precise management of gall bladder disease. This fact is underscored by the recognition that the organ is in juxtaposition to a number of major vascular structures as well as other major viscera of both gastrointestinal and genitourinary tracts. Biliary anomalies are not uncommon, and failure to recognise such a congenital problem may result in significant perioperative morbidity and mortality.

The gall bladder is a sac like hollow organ measuring about 10 cms in length that lies in a fossa on the under surface of the liver. Its position defines the anatomic boundary between the right and left lobes of the liver and division of vascular and ductal system. The gall bladder is attached to the liver by loose areolar tissue rich in small blood vessels and Lymphatics. Extrahepatic portion of the gall bladder is covered by a peritoneum.

The gall bladder typically lies in close proximity to the duodenum, pylorus, hepatic flexure of the right colon and right kidney.

The gall bladder is divided into:-

a. Fundus b. Body c. Infundibulum d. Neck

a. **Fundus**: It is the rounded blind portion of the gall bladder that extends for a short distance beyond the liver edge, and it is the least vascularised, and therefore more susceptible to ischemic changes, including perforation.

b. **Body**: This constitutes the majority of the gall bladder and as such comes in contact with liver, duodenum and hepatic flexure of colon.

c. I**nfundibulum:** Also known as **'Hartmanns'** pouch it is a small bulbous diverticulum, typically lying on the inferior surface of the gall bladder. This anatomic site is clinically significant because stones may become impacted in the infundibulum and obstruct the cystic duct.

d. **Neck** : This is the narrow portion of the gall bladder that lies between the body and cystic duct region.

The Cystic Duct:

This connects the gall bladder to the common bile duct mostly in the supraduodenal part (80%). This is usually 3 cm long with a lumen of about l-3mm. The mucosa of the cystic duct is arranged in spiral folds, known as **the** valves of HEISTER', and its wall is surrounded by a sphincteric structure called the sphincter of 'LUTKINS'.

The importance of the spiral folds of mucosa in the cystic duct from the surgical point of view is that, it may interfere with the passage of an instrument, and also may stop the passage of gall stones.

BLOOD SUPPLY / LYMPHATICS AND INNERVATION:

Arterial supply:

The gall bladder is supplied by the cystic artery which most commonly is a single branch of right hepatic artery. The cystic artery may also originate from the left hepatic, common hepatic, gastroduodenal or superior mesenteric artery. The cystic artery is usually located parallel and medial to the cystic duct, but its course may vary with its origin. The cystic artery divides into superficial and deep branches before entering the gall bladder.

The most dangerous anomalies are when the hepatic artery takes a tortuous course in the front of the origin of the cystic duct, or the right hepatic artery is tortuous and the cystic artery short. This tortuosity is usually known as the "CATERPILLAR TURN' or 'MOYNIHAN'S HUMP'. This variation is the cause of many problems during a difficult cholecystectomy with inflammation in the region of the cystic duct, lnadventant damage to the right hepatic artery in the most difficult to control laparoscopicaly.

Veins

Hepatic surface of the gall bladder is drained **by** numerous small veins passing through the gall bladder bed to break up into capillaries within the liver substance. They do not form a single cystic vein, but the many small veins enter the liver together with ascending veins from the common bile duct.

Lymphatics

The lymphatic vessels of the gall bladder (subserosal and submucosal) drain into cystic lymph node of "LUND' (the sentinel lymph node), which lies in the fork created by the junction of the cystic and common hepatic ducts. Efferents vessels for this lymph node goes to the hilum of the liver and to the coeliac lymph nodes.

Innervation:

Gall bladder is supplied by both sympathetic and parasympathetic nerves. Parasympathetic nerves are derived from the vagus nerve through the coeliac ganglion.

Sympathetic supply comes from the level of T8 and T9, from where the post ganglionic fibers supply the gall bladder along the hepatic artery.

IMPORTANT LAND MARKS:

Triangle of calot: This hepatocystic triangle **is** formed **by** the cystic duct the common hepatic duct **and** under surface of the liver. It is in this area where you will commonly find the cystic artery and the cystic lymph node of hind, and the course of the cystic artery is commonly behind the hepatic duct.

Pathological correlation in Acute cholecystitis:

There are 2 basic types of gall stones:

1. The cholesterol gall stones - predominating in the Western countries constituting about 70%
2. The pigment / brown stones - constitutes the remainder of about 30%, predominantly in the UK and South asian countries like India.

Pathogenesis of cholesterol gall stones:

There are three stages in the gall stone formation

a. Cholesterol supersaturation
b. Nucleation
c. Stone growth

Cholesterol supersaturation:

Cholesterol, insoluble in water, is held in solution by the detergent actions of bile salts & phospholipids with which it form micelle. Bile containing cholesterol stones has an excess of cholesterol relative to bile salts and phospholipids, thus allowing cholesterol crystals to form. Such bile is termed 'supersaturated' or 'Lithogenic'.

There are several important mechanisms in the formation of lithogenic bile, the most important being increased biliary secretion of cholesterol in condition such as:-

1. Obesity
2. High caloric diets
3. Clofibrate therapy
4. increased activity of HMG - COA reductase
5. Pregnancy, etc.

Kinetic balance between nucleating and anti nucleating factors and stone growth:

Bile contains substances which either inhibit (Alipoprotein Al) or promote (Mucin) the growth of cholesterol crystals. Thus the development of cholesterol stones is influenced by the balance between these two kinetic factors.

Other substances which inhibit gall stone formation are an Anionic polypeptide fragment (AFP) and Immunoglobulin A. It is also thought that either absolute or relative decrease in the amount of APF favours nucleation and growth of cholesterol of monohydrate and pigment crystals.

It should be said that the gall bladder also plays and important role in gall stone formation (both cholesterol and pigment stones). It is evident that 85-90% of stone are encountered in this organ rather than bile ducts.

It has been postulated that the gall bladder may alter the physiochemical composition of bile, favouring nucleation and crystal growth by abnormal absorption / secretion, defective surface PH, stasis resulting from impaired gall bladder emptying and stagnation of bile, or by providing essential nucleating factors including mucin, desquaminated cells, bacteria and refluxed intestinal contents.

Gall bladder Mobility and gall stones:

Obviously bile cyrstallisation and crystal aggregation takes time. Therefore stone formation requires that bile must remain with in the gall bladder for sometime, and it can be postulated that the gall bladder of gall 'stone formers [1] will be less active at evacuating its contents. So there is proof that reduced gall bladder emptying also is a key pathogenic factor in cholesterol stone formation.

Pathogenesis of Pigment stones:

Much less is known about the pathophysiology of pigment stones. These stones are typically found in patients with hemolytic disorders or cirrhosis. The absence of bacteria from most patients with black stones suggest that other factors may be operational. The high incidence of pigment gall stones in patients with hemolytic disorder probably results

from excess loads of bilirubin being presented to liver for secretion. The possible explanation for the high incidence of pigment stones in cirrhotics include hypersplenism leading to hemolysis and impaired conjugation of bilirubin, leading to the formation of pigment stones.

Pathological correlation in Acalculous Cholecystits:

It is an unusual, but potentially lethal complication of gall bladder disease. The precise cause and pathogenesis of this disorder is yet to be determined. The estimated incidence is about 3%. It has been reported in critically ill patients in ICU settings, particularly in patients with MOF, following severe trauma or burn, and as a post operative complication of any major procedure. The clinical settings in which acalculous cholescystitis occur has provided important insights into its possible pathogenesis.

Stasis of bile within the gall bladder has long been considered an important factor in the development of this disorder. Biliary spasm can occur as a result of ampullary spasm secondary to narcotics, decreased gall baladdur emptying during periods of prolonged fasting, cystic duct occlusion secondary to edema etc. Moreover altered viscosity as a result of dehydration and multiple transfusion may play a part.

A cascade of events is initiated with release of specific biochemical mediators of inflammatory response. The role of ischemia is speculative, but as the cystic artery is an end artery with little collateral circulation, it may predispose to organ ischemia and gangrene.

The diagnosis of acalculous cholecystitis posses a considerable challenge to the clinician as the patient is most often in an ICU setting.

Early ultra sonography and a high clinical suspicion based on the clinical setting are the key to the diagnosis. Emergency cholecystectomy is warranted in all the patients, but if the patient's general condition is very poor, then a temporary Cholecystostomy may be done to tide over the crisis.

INVESTIGATION

Over the past 15-20 years, advances have been made in diagnostic radiology and interventional endoscopy that allow the precise visualisation and location of gall bladder pathology and the nature of it specific tests are devised to answer specific questions.

a. Abdominal radiography:

Plain x-ray of the abdomen are of limited value in assessing patients with gall stones. Supine and upright films of the abdomen may be useful in excluding other causes of abdominal pain, such as perforated viscus or a bowel obstruction. Presence of significant amounts of calcium within gallstones, which occur in only 15-20% of patients will cause stones to appear as specific objects located in the right upper quadrant on plain x-rays.

It is important to remember that most patients with gallstones will have normal abdominal x-rays. Here are number of other circumstances in which complication may be suggested by specific radiographic findings. These include presence of air within the biliary and outlying is anatomy in patients with cholecystoenteric fistula or air bubbles in the wall of the gall bladder which may present in patients with emphysematous cholecystitis.

b. Oral cholecystography:

It is a relatively simple test for diagnosing gall stones introduced by Graham and Cole in 1924. This test may permit visualisation of gallstones within the gallbladder; the critical function that is assessed is the absorptive ability of the gallbladder.

A radio opaque iodine containing halogenated dye is orally ingested by the patient. The dye is first absorbed by the GIT and extracted in the liver. The liver then excretes the dye into the ductular system and the dye then passes through the cystic duct into the gall bladder. Ultimately, if there are no gall stones and if the GB has normal mucosal function, the dye becomes concentrated through the physiological absorption of water and solutes. A positive study suggestive of gallstones or gall bladder pathology occur when stones are noted as filling defects in a visualized opaque gall bladder, or when the dye is not adequately concentrated and the gall bladder not visualized. When the later occurs a second double dose of contrast is frequently administered. OCG has now generally been replaced by the development and refinement of abdominal ultrasonography

c. Abdominal Ultrasonography:

The basis for this test is the recording of high frequency sound that is reflected off the interfaces that differ in acoustic impedance. This has a sensitivity rate of 98% and specificity rate of 95%.

A number of parameters can be assessed from Ultrasonogram such as :

1. **Gall** bladder size
2. Gall bladder volume
3. Ejection fraction
4. Wall thickness
5. Calculi thickness
6. Number of calculi
7. Diameter of CBD
8. Calculi in CBD
9. Other abdominal pathologies

Cholelithiasis are usually demonstrated as an echogenic foci with distal acoustic shadowing that move on change in position.

d. Biliary scintigraphy:

The intravenous administration of one of a family of technitium 99 nun labelled Imminodiacetic acid radio isotopes has been utilised to image the liver gall bladder and extra hepatic bilary tract. The basis for

this test in the clearance of the isotope from the blood by hepatocytes and its excretion into the bile ducts. This test provides specific information regarding the patency of the cystic duct. In this context it is sensitive in diagnosing patients with acute cholecystitis. Unlike USG which in an anatomic test, this is a functional test. Infact these two are complimentary.

The radio pharmaceuticals used are:

HI DA : Ethyl - hydroxy - imminodiacetic acid.

P1PIDA : Para - isopropyl acitanilido - immino - diacetic acid.

An intravenous injection of 200 mbq is given and the analogue images obtained serially. A normal scan (negative) shows transit from the liver through the biliary tree into the gall bladder and small bowel. Addl. projection (ex. left anterior oblique) can be obtained in doubtful cases.

Failure of scintigraphy to demonstrate any part of the biliary tree indicates hepatic dysfunction or biliary obstruction. The scintigraphy is considered 'positive' if gall bladder is visualised only after 2 hours or is not visualised at all after 24 hours, indicating the possibility of Acute cholecystitis.

Ultrasound Vs. I11DA Scan

Ultrasound though fails to score in the area of specificity it gives more information than HI DA scanning in evaluating the right upper

quadrant pain, since it can pick up other pathologies as well; moreover it can give pre operative evaluation to predict technical difficulties and complications in laparoscopic cholecystectomy.

c. Computerised Tomography:

Unlike ultrasonography CT scan rely on ionising radialion for images. This test is not particularly sensitive for gall stones or **inflammatory** gall bladder disease, but provides much information regarding the nature, extent and location of biliary dilatation, and masses in and around the biliary tract and pancreas.

In general this test provides more useful information when evaluating the jaundiced patients than does ultrasound. Limiting factors includes patient exposure to ionising radiation and cost.

f. Magnetic resonance imaging:

Although this diagnostic test provides images that may appear similar to CT scans the images result from the different magnetic properties of the tissues. A contrast material is usually used for this test - "gadolinium'. This material injected IV. affect the magnetic properties of tissues. Its role in evaluation of patients with hepatobiliary disease remains unclear at this moment of time.

MODALITIES OF MANAGEMENT OF ACUTE CHOLECYSTITIS

A. CHOLECYSTOSTOMY

CHOLECYSTECTOMY IS THE STANDARD TREATMENT FOR ACUTE CHOLECYSTITIS. High risk critically ill patients however often presenting with multisystem disease have increased morbidity and mortality during acute cholecystitis. There are two types of cholecystostomy.

1. Surgical cholecystostomy
2. Percutaneous ultrasonographic guided cholecystostomy

Surgical cholecystostomy:

Surgical cholecystostomy has been advocated for certain high risk groups:

1. Patients with sepsis and failure.
2. Severe cardiac failure
3. Renal failure
4. Hepatic failure
5. Patients in whom dissection of porta is difficult.

CHOLECYSTOSTOMY MINIMISES THE OPERATIVE RISK because it can be performed rapidly with comparatively decreased invasiveness and in some Circumstances under local anesthesia. I however, mortality rates with surgical cholecystostomy was as high as 20-30% usually due to patients underlying disease process (Glenn, Goldmann et al).

The procedure usually used is, under general or regional anesthesia a suitable right sub-costal or paramedian incision is made and the gallbladder drained and stones if any evacuated. Large bore rubber catheter is introduced into the gallbladder and drawn through a separate skin incision.

In the very ill patients with empyema of gall bladder, severe concomitant disease, or sepsis, Cholecystostomy may carry a high operative risk - for these patients a limited procedure is warranted acutely and followed by a definitive procedure later on. Cholecystectomy is a short and simple procedure which could therefore be considered in such circumstances.

PERCUTANEOUS CHOLECYSTOSTOMY

Percutaneous transhepatic cholecystostomy is performed using Hawkins fine needle technique, with placement of accordion catheters into the gallbladder. Under local anesthesia with ultrasonographic or CT guidance a 22 gauge Hawkins needle in advanced through the liver edge into the gall bladder. Bile is aspirated to ensure adequate placement which is confirmed with 1 ml injection of contrast material. A 6.5 fr accordion catheter is advanced into the gall bladder over the needle. The accordion

catheter is retracted to form a T-configuration to prevent catheter dislodgement (Boland et al).

The high risk population who usually undergo guided percutaneous cholecystostomy is as follows:

1. Advanced age
2. Coronary artery disease
3. Sepsis
4. Terminal cancer
5. Uncontrolled IDDM/N1DDM
6. Severe pulmonary disease
7. Immuno suppression
8. Cirrhosis
9. Renal failure
10. Severe mal nutrition
11. ARDS
12. Recent MI
13. Acute pancreatitis
14. Sickle cell crisis

(Suzanne Klimberg et al Univ. of Florido College USA). The clinical criteria for improvement included immediate resolution of

pain - defervescence, decreased WBC, early improvement in liver function profile.

Percutaneous cholecystostomy has previously been documented and affords advantages over surgical therapy in selected patients. The procedure can readily be performed on emergency basis under local anesthesia often without moving the patients, it requires only minutes to complete insertion, allows postponement of the definitive procedure until the disease or sepsis well controlled and has the advantage of being diagnostic and therapeutic.

B. CHOLECYSTECTOMY

1. a. EMERGENCY /EARLY CHOLECYSTECTOMY
   b. DELAYED CHOLECYSTECTOMY
2. OPEN CHOLECYSTECTOMY
3. LAPAROSCOPIC CHOLECYSTECTOMY
4. MINI LAP CHOLECYSTECTOMY
5. MINIMALLY INVASIVE CHOLECYSTECTOMY

CHOLECYSTECTOMY IS THE GOLD STANDARD FOR THE TREATMENT OF ACUTE CHOLECYSTITIS. Previously majority of the general surgeons treated cholecystitis conservatively with intravenous fluids and analgesics in conjunction with antibiotics. Delayed cholecystectomy is carried out during a second admission after the acute attack has settled down.

EMERGENCY CHOLECYSTECTOMY

Emergency cholecystectomy is perfonned immediately on an emergency basis if the patients condition is deteriorating during 24-48 hrs.

EARLY CHOLECYSTECTOMY

In all but the emergency cases patients can be taken up for the surgery on the next available list.

Cholecystectomy can be performed through a paramedian incision or kocher's subcostal incision. Pathological state of the gall bladder is classified as uncomplicated when features of simple inflammed gall bladder alone is present as acute cholecystitis or complicated when empyema, mucocele, gangrenous gall bladder or frank perforation a present.

However, in acute cholecystitis the gall bladder wall is thickened and edematous, and the edema is responsible for most of the wall thickening. Although the serous surface is congested and covered with a fibrinous exudate, this is not purulent, polymorphs and bacteri are few, and the inflammatory exudate is more marked in the outer layers of the wall. It is this outer wall edema that provides a plane of cleavage between liver and the gall bladder, facilitating enucleation and making cholecystectomy less difficult in the acute stage rather than when this phase has settled down with the formation of postinfective fibrous tissues.

Cholecystectomy can be done by fundus first or cystic duct first technique. The fundus first technique is especially advocated for mucocele or empyema. When the gall bladder is distended with acute inflammation of the wall, the associated adhesions to the stomach and colon and often a localised abscess it can be difficult to isolate the cystic duct, common duct junction and' cystic artery as a preliminary procedure. It is believed that fundus first cholecystectomy can be the only safe way to remove an acutely inflammed gall bladder in order to obviate the risk of damage to CBD, sometimes the cystic duct first technique may have to be abandoned and converted to fundus first technique in certain difficult cases.

C. LAPAROSCOPIC CHOLECYSTECTOMY

Since the operation was first performed in man by Philip Mouret in Lyon in 1987, Laparoscopic cholecystectomy has become firmly established in number of centres all over USA, UK and not forgetting India.

The essential attributes of the laparoscopic approach, which is as invasive as the equivalent open operation is the reduction of trauma of access without compromise of the exposure of the operative field.

Patients were operated under general anesthesia in supine position with the surgeon on the left side and assistant on the right side (the DUNDEE Technique) using TV monitors.

LAPAROSCOPIC CHOLECYSTECTOMY

CREATION OF PNEUMOPERITONEUM

Thinnest area of the anterior abdominal wall is at the umbilicus making it most safe for insertion of insufllation needle. The alternate sites are left or right midclavicular line just below costal margin.

PRIMARY PORTAL

Primary trocar is inserted through a skin incision in the lower aspect of umbilicus. The umbilical site is preferred when performing cholecystectomy because single layer of fascia and no muscle layer in this area facilitates removal of organ easily.

ACCESSORY PORTALS

After insufflation and insertion of the umbilical trocar, the laparoscope is inserted and turned cephalad. Secondary 5mm trocars are inserted in the midclavicular line two linger widths below right costal margin or in the anterior axillary line 2 fingers breadth below costal margin.

MANIPULATION OF GALL BLADDER

Atraumatic grasping forceps are introduced through the accesory lateral portal. The lateral forceps grasps the fundus of the gall bladder and lifts and pushes it cephalad over the edge of the liver. This retracts the liver superiorly and exposes Hartmann's pouch. Another grasping forceps inserted into the midclavicular accessory portal. This instrument grasps the pouch and pulls it laterally placing torsion on the cystic duct and common bile duct.

DISSECTING CYSTIC DUCT AND ARTERY

Dissection is carried out almost entirely from the accessory portal. Blunt forceps are utilised to dissect the peritoneal attachments of hepatoduodenal lingament from the cystic duct and artery. Dissection is performed from gall bladder down towards CBD. Should avulsion of artery occur this technique would leave a long stump to retrieve. Cystic artery should be clamped and transected prior to cystic duct.

DISSECTING GALL BLADDER FROM FOSSA

The neck of the gallbladder is retracted laterally by forceps placed through midclavicular portal. Cautery is introduced through the medial portal and cystic duct and artery are transected. The midclavicular forceps

pulls the neck of the gallbladder superiorly to laterally while superior traction is placed on fundus. The last centimeter of the gallbladder near the funduns is the most difficult because there is not good traction.

REMOVING GALLBLADDER

The easiest route of removal is through the umbilicus since there are no muscle layers and only on fascial plane. As and when neck is exposed a small incision made on the neck of the gallbladder and decompressed with suction. The gallbladder then removed.

REASONS FOR CONVERSION FROM LAPAROSCOPIC TO OPEN CHOLECYSTECTOMY

Although laparoscopic cholecystectomy can be successfully performed majority of the time there remains a significant number of patients who require conversion to open cholecystectomy. A study conducted for this purpose by Jeffrey H., Peters et al (AJ SURGERY Dec 1998) outlined the common reasons for conversions, they are:

I. DIFFICULT DISSECTION

- Dense adhesions
- Severe inflammation
- Obscure anatomy
- Retraction difficulty

II. COMMON BILE DUCT

- Abnormal laparoscopic intraoperative cholangiogram
- failed attempt at CBD exploration

  failed intraoperative cholangiogram

III. COMPLICATIONS

- Bleeding
- Duodenal injury
- Cystic duct avulsion
- Respiratory acidosis

It was shown that patients presenting with symptoms of radiographic findings of acute cholecystitis are at significant risk for conversion to laparotomy. Difficult dissection, usually secondary to adhesions, severe inflammation or obscure anatomy is the most common reason for conversion.

MINI - LAP CHOLECYSTECTOMY

This is done through a small subcostal approach. A 6-8 cm incision is made in the subcostal line and the peritoneum opened and routine cholecystectomy performed. Advantages are small scar, less postoperative discomfort (Calhoun et al).

MINIMALLY INVASIVE CHOLECYSTECTOMY

Narendra Tyagi et al at St. Joseph Mercy Hospital, Pontiac, Michigan has advocated minimally invasive technique for cholecystectomy via microceliotomy through 3 cm transverse high sub

xiphoid incision in the MINIMAL STRESS TRIANGLE (MST). MST is formed by the medial margins of the sixth to eighth costochondral cartilage as the lateral sides of the triangle within the epigastric area. The base of the triangle is formed by a plane joining both the bilateral eighth costal cartilage. The Calots triangle lies within the boundaries ofMST. ENDOSCOPIC INSTRUMENTS ARE USED FOR SURGERY. Entry into the abdominal cavity is made through the falciform ligament. Entry through the falciform ligament is associated with minimum disruption of the parietal peritoneum. MICROCELIOTOMY was performed under direct vision without the need of pneumoperitoneum or video equipment.

Microceliotomy is said to offer numerous advantages. A minimally -invasive procedure, rapid post operative recovery, acceptability of scar size, early to return to work and reduced post surgical analgesic requirements.

AIMS AND OBJECTIVES

1. To study the incidence of acute cholecystitis in different age group in both sexes.

2. To study the different causes of acute cholecystitis.

3. To diagnose acute cholecystitis with the help of clinical features and different modalities of investigations.

4. Management of acute cholecystitis.

5. To study the complications, management of complications and follow up.

MATERIALS AND METHODS

50 cases diagnosed as acute cholecystitis were entered into the study by any one or more of the following methods form Govt. K.A.P Vishwanathan Medical College and Hospital, Trichirappalli June 2008 to Oct 2009.

CLINICAL FEATURES

1. Right upper quadrant pain similar to biliary colic in onset and character but persists beyond 4-6 hrs.
2. Associated anorexia, nausea and vomiting.
3. Low grade fever.
4. Localised right upper quadrant tenderness.
5. MURPHY'S SIGN positive.
6. Presence of inflammatory mass in right upper quadrant.
7. BOAS'S SIGN POSITIVE - Hyperaesthesia in the right posterior 9-11 * intercostal space.

INVESTIGATIONS

1. **PBS - Peripheral blood smear**
   - to diagnose raised WBC count
   - 12000 - 15000 indicate acute cholecystitis
   - More than 20,000 indicate complications such as perforation and gangrene.

2. Biochemical evaluation :-

a. Serum electrolytes - To diagnose co-existent electrolyte abnormalities if vomiting is present.

b. Blood Urea level - To diagnose renal failure if it occur asa component of multiorgan failure if sepsis ensues and as a routine preoperative evaluation.

c. Blood sugar level - As a routine investigation and to identify diabetic patients who are at a high risk of gangrene

and perforation of gall bladder.

d. Liver function tests:-

- To identify deranged liver metabolism secondary to acute cholecystitis and sepsis.
- In case of biliary outflow obstruction like carcinoma pancreas which cause early obstructive jaundice with elevated alkaline phosphatase.

e. E.T. Prothrombine time:-

It is the earliest indicator of liver cell failure.

3. **PLAIN X-RAY ABDOMEN**

Patients were subjected to routine plain X-Ray Abdomen to rule out other causes of acute abdomen like

a. Duodenal ulcer with gas under the diaphragm
b. Calculi in upper renal system
c. Gas in the biliary system,

and to pick up radiopaque stones if any.

4. ULTRASOUND OF ABDOMEN

Patients were subjected to ultrasound evaluation on clinical suspicion of acute cholecystitis.

The following findings, if present were suggestive ***o f acute,*** cholecystitis

1. Presence of calculi
2. Presence of gall bladder wall edema >3mm
3. pericholecystic collection
4. Evidence of free fluid in the subhepatic space
5. Presence of calculi obstructing the cystic duct.
6. Presence of adhesions if any
7. Presence of sonographic Murphy's sign

MANAGEMENT

Patients who were diagnosed as acute cholecystitis were subjected to cholecystostomy or cholecystectomy according to the situation.

CHOLECYSTOSTOMY - This is done after informed consent, if the general condition of the patient does not permit for cholecystectomy.

EMERGENCY CHOLECYSTECTOMY - This is done after informed consent in an emergency basis if the patient after the usual management with I.V. fluids, nasogastric suction does not settle and

proceeds a downhill course. Patients were taken up for surgery immediately within 4-6 hrs.

EARLY CHOLECYSTECTOMY - Patients of acute cholecystitis who seem to settle with IV fluids, antibiotics and nasogastric suction were operated on the next operative list after prior consent.

ELECTIVE CHOLECYSTECTOMY - Patients were discharged home after the acute attack for 4-6 weeks and called back for elective cholecystectomy.

LAPAROSCOPIC CHOLECYSTECTOMY - Consent taken from the patient regarding the procedure explaining the advantages and disadvantages.

DRAINS

A Standard Suction drain was used for all open cholecystectomies. Drains were removed from 3rd - 5th day.

OBSERVATIONS

50 cases of Acute cholecystitis was studied in the given period of June 2007 to June 2009. The results were tabulated as follows:-

TYPES OF CASES STUDIES

Type of Pathology	No. of Cases	Percentage (%)
A. CALCULOUS CHOLECYSTITIS	42	84
B. ACALCULOUS CHOLECYSTITIS		
Malignancy	1	
Idiopathic acalculous cholecystitis	7	14

AGE DISTRIBUTION

Age	Calculous Cholecystitis	Acalculous Cholecystitis	Percentage (%)
1. 20-25 Yrs	3	1	8
2. 25-30 Yrs	2	0	4
3. 30-35 Yrs	5	0	10
4. 35-40 Yrs	3	1	8
5. 40-45 Yrs	6	2	16
6. 45-50 yrs	13	1	28
7. 50 & above	10	3	26

SEX DISTRIBUTION

Sex	Calculous Cholecystitis	Acalculous Cholecystitis	Total	Percentage **(%)**
Male	19	3	22	44
Female	23	5	28	56

MODES OF CLINICAL PRESENTATION

	Number of Patients	Percentage
Right Hypo Chondral Tenderness	50	100%
Fever	23	46%
Vomiting	12	24%
Jaundice	3	6%
Leucocytosis	17	34%

MANAGEMENT GIVEN

Management	Calculous Cholecystitis	Acalculous Cholecystitis	Total	Percen tage (%)
Emergency cholecystectomy	4	2	6	12 -
Early cholecystectomy	24.	3	27	54
Elective cholecystectomy	14		16	32
Cholecystostomy	0	1	1	2
Laparscopic Cholecystectomy	0	0	0	0

TYPES OF ORGANISM CULCUTRED FROM BILE

Organism Cultured	Calculous cholecystitis	Acalculous Cholecystitis	Percent Age (%)
E.Coli	10	2	24
Staph, aureus	7	0	14
Salmonella	1	0	2
NO organisms	24	6	60

ORGANISMS CULTURED VS. WOUND INFECTION

Organisms Cultured	% Culture Positive	Wound Infection Cases	Percentage (%)
E.Coli	24	2	4
Staph. Aureus	14	2	4
Salmonella	2	1	2
No. organisms	60	4	8
		Total	**18**

WOULD INFECTION RATE

Type of Surgery	Total Patient	Wound Infection	Percentage (%)
Emergency	6	1	19
Elective	44	8	17.7

DISCUSSION

50 cases of Acute cholecystitis were studied in Government K.A.P Vishwanathan Medical College Hospital the study period of 3 years.

Acute cholecystitis is a frequent cause of abdominal emergencies. It is an acute upper abdominal condition always associated with macroscopic and microscopic acute inflammatory changes in the gallbladder.

The diagnosis of acute cholecystitis is established by the following factors.

1. Right upper quadrant pain similar to biliary colic in onset and character but persists beyond 4-6 hrs.
2. Associated anorexia, nausea, vomiting.
3. Low grade fever.
4. Localised Right upper quadrant tenderness
5. Presence of guarding and rebound tenderness
6. MURPHY'S SIGN positive – Inspiratory arrest during deep palpation in right upper quadrant.
7. BOAS'S SIGN – Hyperaesthesia over 9^{th} – 11 Intercostal space posteriorly on the right side.
8. Presence of a mass in right upper quadrant.

9. Leukocytosis of 12,000 – 15,000.
10. X-ray Abdomen showing calculi, gas in the biliary tract.
11. Ultrasound evaluation.
12. Non-visualisation of gallbladder in 99m Technetium IDA Scintigraphy.

AGE DISTRIBUTION

In our study population of 50 cases maximum incidence occurred in the subgroup of population in 45-50 vrs. This is in correlation with earlier literature which says acute cholecystitis is a disease of middle age, mainly calculous type. The another group is the malignancy group, which becomes more common in the older category. 1 case was found to be due to malignancy causing obstructive jaundice with cholecystitis. This was treated with cholecystectomy initially and was later bypassed with cholecystectomy which totally ameliorated the symptoms & jaundice. The older saying of gallstone disease occurs in the 'fat, fertile, female of forty' was not found to be 100% true. It emphasizes the point of gallstones to be common in well nourished women of middle age group. This common prevalence of gallstones in the middle age group leads to the increased prevalence of Acute cholecystitis in the middle age group.

This was reflected even in our study of 50 cases which showed a prevalence of 28% of cases in 45-50 yrs. group.

Surprisingly acute cholecystitis in the paediatric age group was not encountered at all. This is because gallstones are relatively rare in otherwise healthy children, occurring more common in patients with predisposing factors.

The predisposing causes in children are hemolytic disorders, (Sickle cell disease, thalassemia, red cell enzymopathy, obesity ileal resection). They have more of pigment stones, rather than cholesterol stones. Since presence of stones itself is rare the condition of acute cholecystitis is still rare an entity. Even in pediatric age group the treatment of calculous cholecystitis is cholecystectomy. Laparoscopic cholecystectomy is also possible in pediatric age group. (Ware Kinney ***et al*** J. Pediat. 1997). Acalculous disease is very uncommon may occur with similar pattern as adults and also with infections particularly Streptococci A,B, Salmonella and Leptospirosis. Our study identified one case of calculous cholecystitis with salmonella infection on bile culture but the age was in older group of 55 yrs.

Acalculous cholecystitis was found in the more older group of patients. 8 cases of acalculous cholecystitis was found amounting to 16%

of cases. Out of which 1 was malignancy case causing biliary outflow obstruction and cholecystitis. This is concomitant with study by Shapiro ***et al*** mentioning an incidence of 2 - 15% of cases of acute cholecystitis.

SEX DISTRIBUTION

Out of the 50 cases studied 28 were females and 22 were males accounting to 56% and 44% respectively. It showed that incidence of acute cholecystitis was higher in female population than the males. Majority of the females were married and had more than one children. This is consistent with literature that acute cholecystitis is common surgical emergency in the middle aged women (Schwartz). There are many reasons that the female population is a target for this disease. 23 patients had calculous cholecystitis and 5 patients had acalculous cholecystitis. As mentioned previously cholecystitis is common among middle aged women. The gallstones were more common in the multiparous women, Barbara ***et al.*** The central mechanism in the formation of gallstones is gallbladder dysmotility. Stone formation requires that bile must remain within the gallbladder for sometime and it can be postulated that the gallbladder of gallstone-formers will be less active in evacuating the contents (Everson ***et al.*** Therefore gallbladder emptying is a key pathogenic defect in stone formation apart from bile supersaturation, chemistry of bile for crystal promotion and aggregation.

It was found that during pregnancy there is impairment of gallbladder motility (Everson ***et at)*** Ryan ***et al*** at their study proved a direct progestagenic effect on gallbladder muscle receptors. This causes impaired motility causing sludging and later crystallization, stone formation, obstruction and cholecystitis. This may be possible explanation of the double fold increase in the incidence of Acute cholecystitis in women.

8 patients had acalculous cholecystitis. All were acute cholecystitis with gallbladder edema, but no demonstrable stone radiologically and sm^iealJY-r-^ Histology showed acute inflammation of the gallbladder. No predisposing factors of critical illness, post-surgery, trauma, burns were identified. Patients presented with RUQ tenderness guarding, mild pyrexia and vomiting. No stone was demonstrated intraoperatively. Two patients were multiparous. One patient was in the post partum period - 2 weeks after delivery. These cases could be explained by the gallbladder dysmotility seen during pregnancy (Ryan *et al).*

ACUTE CALCULOUS OR ACALCULOUS CHOLECYSTITIS

Out of the 50 cases studied 42 of them were of Acute calculous type and 8 cases were of acalculous type, which amounts to 84% and 16% respectively.

SI. No.	Age	Calculous Cholecystitis	Acalculous Cholecystitis	Percentage (%)
1.	20-25 Yrs	3	1	8
2.	25-30 Yrs	2	0	4
3.	30-35 Yrs	5	0	10
4.	35-40 Yrs	3	1	8
5.	40-45 Yrs	6	2	16
6.	45-59 Yrs	13	1	28
7.	50 & above	10	3	26

Calculous cholecystitis 84% of cases. Acalculous cholecystitis 16% of cases.

Kune and Birks study of 1970 showed gallstones in 98% of acute cholecystitis, malignant obstruction in 1% and acalculous cholecystitis 1%.

In out study malignant obstruction is included in the acalculous type.

	Calculous	Acalculous
Kurne, Birks	98%	2%
Shapiro et al	85%	15%
Present study	84%	16%

The 8 cases of acalculous variety were diagnosed with the aid of Ultrasonography. They showed gallbladder wall thickness more than 7mm with edema and pejjcholecystic collection. No evidence of stone was found in the gallbladder of the CBD.

Savaco ***et al*** 1996 in their study in Yale University studied the de novo presentation of acute acalculous cholecystitis in the outpatients. In their study 47 patients were studied of which 36 patients (77%) did not have any evidence of acute illness or trauma. The study showed that acalculous cholecystitis can occur de novo without the setting of Intensive care surroundings of narcotics, ventilation, TPN or Transfusions. 3 patients in our study were males. In the Yale study significant vascular disease was observed in 72% of the patients. The 3 patients in our study did not have any evidence of vascular disease. The other 5 patients were females who were multiparous and non-obese. No risk factors were found.

MODES OF CLINICAL PRESENTATION

In our study of 50 patients, all patients (50) presented with right hypochondrial tenderness. 46% (23 patients) presented with pyrexia. Anorexia or vomiting was observed in 24%, leucocytosis in 34%.

Jaundice was present in 3 patients, of which 2 had CBD stones, they underwent CBD exploration. 1 patient had carcinoma Gall bladder and that patient underwent cholecystostomy.

DIAGNOSIS BY ULTRASONOGRAPHY

50 cases were evaluated ultrasonographically. Once the suspicion of cholecystitis occurred they were subjected to sonography. The findings are tabulated as below:

Parameter	Variables	Patients
Gallbladder size	Normal	20
	Contracted	4
	Distended	25
Wall thickness	<5 mm	2
	5-7 mm	22
	>7 mm	26
Number of calculi	Single	5
	Multiple	37

Abnormally in several different right quadrants organs, including liver, gallbladder, biliary tree, pancreas, right kidney and duodenum may cause similar pain patterns and clinical findings. In addition in the gallbladder, the different complications resulting from the presence of gallstone namely acute, subacute and chronic cholecystitis and biliary colic all may produce similar type of right upper quadrant pain. Acute cholecystitis itself may produce severe symptoms in some patients but may be less symptomatic in others particularly elderly. Because of this, all patients with right upper quadrant pain should be suspected with some degree of suspicion for acute cholecystitis.

Ultrasound can predict the difficulty and complications during laparoscopy (Peter Corr) ***et al*** A.IS. The most valuable assessments ultrasound can provide are gallbladder wall thickness, gallbladder function, gallstone size, CBD diameter and gallbladder ejection fraction. Significant association was found between diminished gallbladder function, wall thickening and increasing difficulty in laparoscopic surgery. In our study all patients operated had positive ultrasound findings upto 100% as mentioned in the above table. If ultrasound is not helpful then IDA scintigraphy could be done to rule out Acute cholecystitis. Negative IDA scan rules out Acute cholecystitis but positive reports should be interpreted with caution.

CHOLECYSTECTOMY, CHOLECYSTOSTOMY

In our 50 cases studied 6 patients underwent emergency cholecystectomy 12% out of which 4 were for Calculous cholecystitis and 2 patients for acalculous cholecystitis. There were 4 gangrenous gallbaladder - 3 in calculous and 1 in acaculous group. No mortality was encountered in any of these.

It is advisable to do emergency/early cholecystectomy in acute cholecystitis because 22-27% of the patients who are sent home to await surgery will be readmitted with further attack in the waiting period. (Van Rensburg. BJS).

Norrby ***et al*** in a prospective randomised trial, showed that there was no difference in the frequency of intra-and post operative complications and the incidence of bacterial complications was markedly lower in the early group although a moderate but significantly greater intra operative blood loss was reported. In acute cholecystitis the advantage in emergency/early cholecystectomy was mentioned by (Addison ***et at).*** It is said the outer wall edema provides a plane of cleavage between liver and gallbladder facilitating enucleation and making cholecystectomy less difficult in acute stage rather than when this phase has settled down with formation of post infective fibrous tissue. Malinovski ***et al.*** has showed emergency cholecystectomy is the treatment of choice even in patients over 70 yrs. of age.In our study only 2 such elderly patients were encountered. No laparoscopic cholecy stectomies were done in our study. Velasco ***et al*** have shown that laparoscopic cholecystectomy can be done on an emergent basis with similar results as of open cholecystectomy.

Early cholecystectomy was done in 27 cases with no mortality. World wide mortality rate for cholecystectomy is 0.5% (Addison ***et at).*** In our study cholecystectomy patients had no mortality.

Elective cholecystectomy was done on 16 patients. The post operative period was more comfortable with less analgesic use with reduced patient discomfot. Minimally invasive surgery is at present an alternative approach to diminish metabolic response by avoiding a substantial incision. Study conducted by Gallaghar ***et al*** showed improved respiratory function during the post operative period.

Cholecystostomy was done for 1 patient with malignant obstruction of biliary tract. The patient came with obstructive jaundice with bilirubin in the level of 15 mg/dl and above. Cholecystectomy is the ultimate solution to acute cholecystitis. In the very ill patient with severe concomitant disease, malignancy or sepsis a limited procedure is warranted. Winkler, Kaplan ***et al*** has showed only a 5% mortality with cholecystomy in high risk patients, but our patient ultimately succumbed to the disease.

BILE CULTURE

Out of the 50 cases studied 20 cases were bile culture positive for microorganisms. This accounts for 40% of the cases. Bile culture showed no growth in 60% of the cases 12 cases were positive for E. coli infection. In our study this was the commonest organism cultured. Next in row comes staphylococcus aureus. 7 cases were positive for S. aureus. One

unique case was positive for Salmonella typhi. Patient was subsequently treated with appropriate antibiotic.

BILE CULTURE

	Fukunagal *et al*	**Famel *et al***	**Our Study**
E. coli	32%	42%	24%
Staphi	25.2	23%	14%
No organism	31%	20	60%

Lykkegaud and Nielson showed that bile in the nondiseased biliary tract is sterile. As one encounters more complex biliary tract disease, the incidence of positive culture increases. There are three theories for pathogenesis of bactibilia.

1. Enterohepatic route
2. Ascending route
3. Hematogenous route

ENTERO HEPATIC ROUTE, in which colonic organisms travel via the portal blood to the liver are excreted in the bile and proliferate in the stagnant bile of diseased billy tract.

THE ASCENDING ROUTE, in which organisms appear form duodenum, traverse Vater's papilla and ascend the biliary tree.

HEMATOGENOUS ROUTE in which blood organisms are carried passively to the diseased biliary tract where it proliferates.

As said before some element of obstruction and stagnation is absolutely necessary for the growth of organisms. Growth of the organism is not the primary pathology as said in the review earlier in the case Acute cholecystitis. It is the obstruction and release of inflammatory mediators with vascular obstruction and ischemia that initiates the damage and later making way for the colonization of the bacteria.

Apart from E.coli & Staph aureus the other pathogens sometimes identified are klebsiella, Streptococcus viridans, Clostridium perfringens, Lactobacillus, Enterobacter, Proteus, Citrobacter and bacteroides (Pott ***et al.,*** Fukunage ***et al*** and Famelle ***et al).***

DRAINAGE OF GALLBLADDER BED

The great majority of surgeons routinely place a drain down to the gallbladder bed following cholecystectomy. The rationale is that drain will allow the escape of any collection of blood from oozing gallbladder bed or collection of bile from an overlooked biliary leakage from the same source or from a slipped cystic duct ligature.

There are two great saying one "against" and one "for" the drains. They are, "THE CEMETERIES ARE FILLED WITH PATIENTS

WHOSE GALLBLADDERS WERE REMOVED WITHOUT DRAINAGE". John B. Draver.

The second saying is a follows.

BILE IS NOT EDUCATED TO CLIMB DRAINS. Frederick Coller.

Yates performed a series of experiments in dogs and concluded that external drainage of the peritoneal Cavity is useless, the peritoneal end of the drain being walled off within a short time. Even today drainage after cholecystectomy has both disciples and dissenters.

There are many studies in the literature that reported random control clinical trials atleast five, but they all did not reach same conclusions, 3 advising drains and 2 not advising drains. But later Playfort ***et al*** (Br.J.Surgery) concluded that drainage or non-drainage of the gallbladder must remain a matter of individual preference.

In our study all patients who underwent open cholecystectomy were put under drain for 3-5 days, followed by removal on the 3rd-5th day. This was the standard protocol followed. Drains used were Redivac suction drain. All drains stopped functioning after 48 hrs. No patient had prolonged biliary drainage. No complications were found.

WALTMAN - WALTERS SYNDROME:

In out study using drains for all cases of open cholecystectomy did not show any complication attributable only to the drain. All patients were put under drain because most of them were operated on an emergency /early basis. Even patients who were operated electively were put on drain. No patients underwent prolonged drainage.

Accumulation of bile in the right sub-phrenic or subhepatic region can occur even when provision for drainage is there. Upper abdominal or chest pain associated with tachycardia and persistent low blood pressure are the cardinal features. This complication if detected early can be cured just by draining of the collection, otherwise rapid downcourse ensues. Such a collection or the complication was not found in our study.

WOUND INFECTION

Out of 50 cases studied bile culture was positive in 40% of the cases (20 cases). No organism was cultured in 60% of the cases. Out of that 9 patients had would infections in the post-operative period which led to gaping of the wound and warranting secondary suturing later on.

Organisms cultured	% Culture positive	Wound infection	% of wound infection
E. coli	24%	2	4%
Staphi	14%	2	4%
Salmonella	2%	1	2%
No organism	2%	4	8%

Four patients developed wound infection in the group where no organisms were cultured. Whereas totally 5 patients developed wound infection when the bile was showing positive culture to organisms, accounting to 10%

The patient who had salmonella growth from the bile had severe wound infection which led to complete breaking of the operative would warranting a secondary suture 10 days later after granulation. It had staph aureus grown from the wound site. In fact our bile culture and wound site culture did not show similar organism but all had staph aureus.

Chetlin and Eliott indicated that the incidence of infection is 40 times greater in patients undergoing biliary tract procedures in the presence of bactibilla. Keighley et al found that patients with bactibilla has an infection rate of 30%.

They also identified high risk groups in which baclibilla exceed 50% They are.

a. acute cholecystitis

b. obstructive jaundice

c. CBD stones.

The elderly patient with acute cholecystitis is more likely to have bacteria in the bile. Fukunage ***et al*** found bile cultures positive in 69% of the patients. In our study we had 40% of patients positive for bileculture. Totally 9 patients had wound infection amounting 18%. In our study group all the patents were on antibiotic cover during surgery.

Richard Garibaldi ***et al*** conducted and extensive study on post cholecystectomy wound infections and concluded that patients who had positive bile cultures taken during surgery or positive intraoperative wound cultures had higher rate of infection than patients with negative cultures. The almost reflected in out study. Which showed that would infection rate is higher in the bile culture positive group.

Richard Garibaldi ***et al*** study showed that the post cholecystectomy wound infection rate was 11 %. Our study reflected a percentage of 18% which is almost similar.

In our study wound infection rale was higher in cases operated on an emergency basts (20%) rather than elective cholecystectomies (18%).

SUMMARY AND CONCLUSIONS

50 cases of acute cholecystitis were studied. Their agewise and sex incidence in the A.G.M. Government Hospital Tiruchirapalli was studied. The different causes of acute cholecystitis were evaluated. The different modalities of diagnosis and investigations were scrutinised. Various ways of management of acute cholecystitis were studied. The incidence of complications were observed. The following is the conclusion of the study.

1. AGE DISTRIBUTION

- Maximum incidence of acute cholecystitis occurred in 45 - 50 age group.
- Gallstone incidence was maximum in the middle age group 45 - 50 yrs. (28%)
- No paediatric case of acute cholecystitis was found.

2. SEX DISTRIBUTION

- Maximum incidence of acute cholecystitis occurred in the female population of 56%
- Multiparous women were more prone for acute cholecystitis.

3. CALCULOUS OR ACALCULOUS CHOLECYSTITIS

- Acute calculous cholecystitis was the commonest type with incidence of 84%
- Acalculous cholecystitis was found to be 16%
- There was no predisposing factors of acute acalculoous cholecystitis demonstrated in our study.

DIAGNOSIS

- Diagnosis was made by clinical suspicion of signs or symptoms.
- Ultrasonographh was positive in all the patients and correlated with pathological conclusion of acute cholecystitis.

MANAGEMENT

CHOLECYSTECTOMY:

- 12% of the patient underwant emergency cholecystectomy with no mortality.
- Early cholecystectomy was done in 54% with no mortality and morbidity significantly less and providing to be superior mode of management.
- Our study had no Laparoscopic cholecystotomy.

CHOLECYSTOSTOMY

- 2% of (he patients underwent cholecystostomy because their general condition did not permit cholecystectomy.

BILE CULTURE AND WOULD INFECTION

- Bile culture was positive in 40% of the patients.
- Wound infection rate was more in the bile culture positive group 10%
- Wound infection was less in the patients who had no organisms cultured from bile - 8%.

DRAINS

All the patients who underwent open cholecystectomy were put on a drain in the sub - hepatic space for 3 - 4 days. No increase in the morbidity or infection was attributed only to the use of drain. Drainage is still under individual preference.

PROFORMA

Name : Age / Sex I P No. Occupation

Address:

Income :

Presenting Complaints - Right upper quadrant pain
Biliary colic
Nausea / Vomitting
Fever, with chills
Jaundice

Past History - Jaundice
Biliary Colic
Previous
Surgeries
DM/HTN/TB/
COPD

Personal History - Oral contraceptive usage
Smoking / Alcohol

ON EXAMINATION

Vital Signs - PR, BP, Temp.

Other Systems	-	CVS/RS/CNS
Per abdomen	-	Guarding/rigidity/
		Right hypochondrium tenderness
		Murphy's sign
	-	Boas' sign
		Right hypochondriac mass abdomen
	-	Cholecystectomy / Cholecystectomy / Lap. Cholecystostomy
Intra-operative findings (Elective/emergency /	-	Gall bladder status
Early/	-	Calot's triangle anatomy
Laparoscopic	-	CBD size, stones
	-	Others
	-	Number and type of stones
	-	Whether drain placed
Post operative course	-	Drain removal
	-	Suture Removal
	-	Wound infection
	-	Wound infection
	-	Respiratory complications
	-	Bile leak
	-	Wattman - Walters Syndrome
	-	Stone analysis
	-	GB Histology

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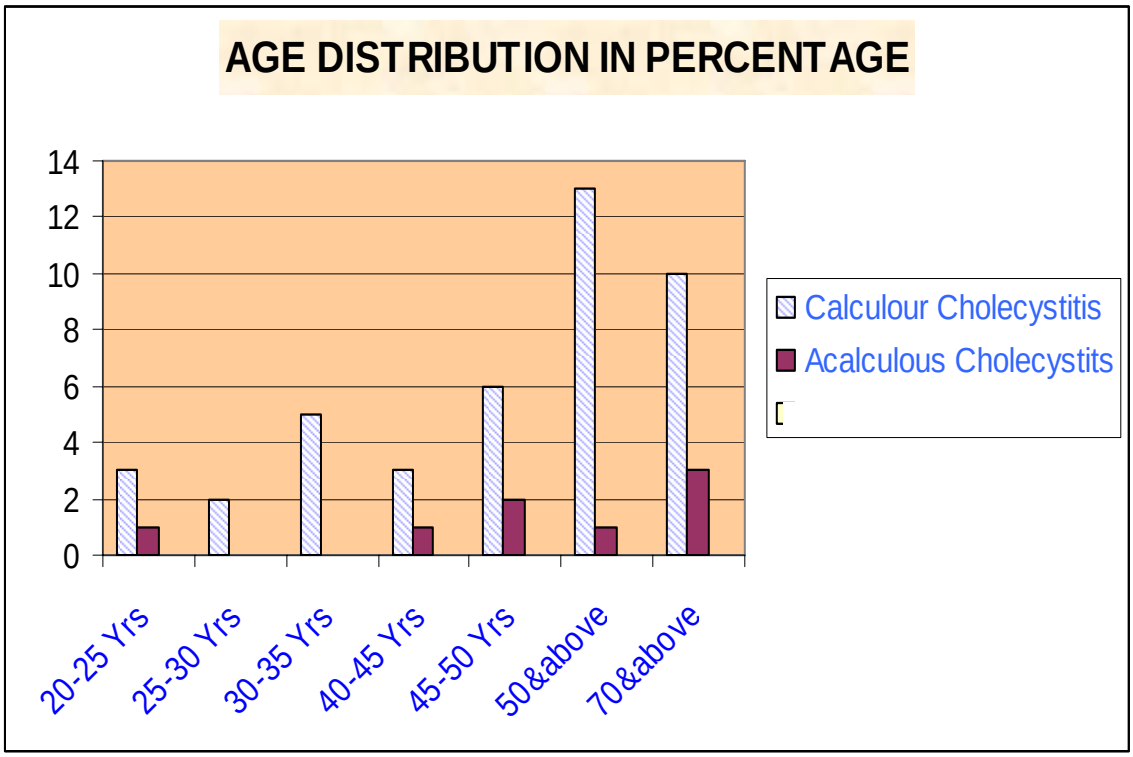
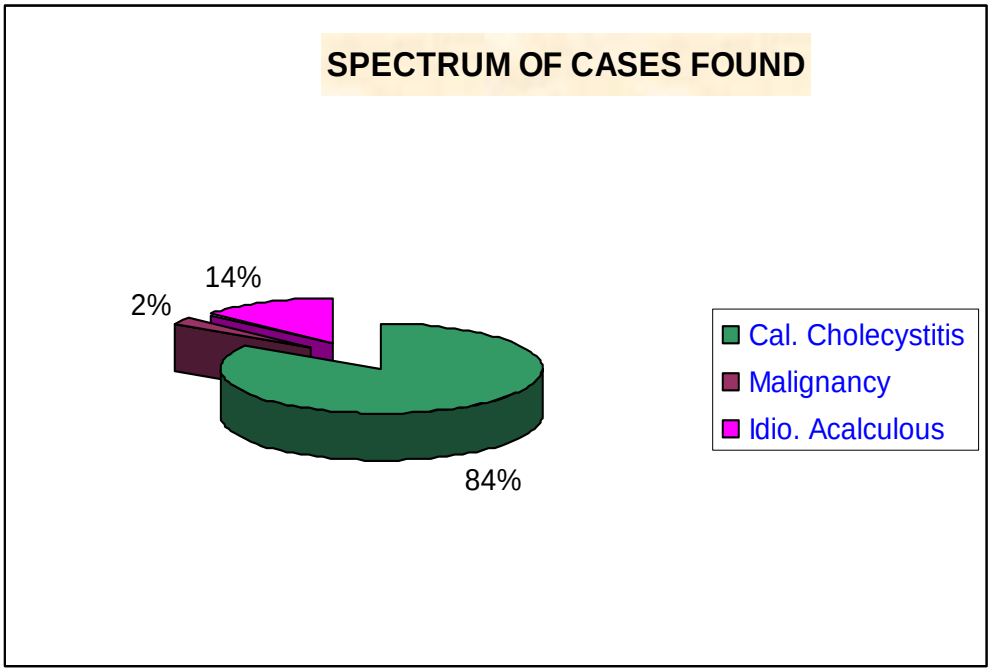

AGE DISTRIBUTION IN PERCENTAGE
14
12
10
8
6
4
2
0
20-25 Yrs
25-30 Yrs
30-35 Yrs
40-45 Yrs
45-50 Yrs
50&above
70&above
Calculour Cholecystitis
Acalculous Cholecystits


SPECTRUM OF CASES FOUND
14%
2%
84%
Cal. Cholecystitis
Malignancy
Idio. Acalculous

MASTER CHART

Sl. No	Name	IP No	Age	Sex	Rt. Hypo pain	Fever	Vomiting	Jaundice	Leuco-cytosis	Type of Chole-cytitis	Bile Organism	Type of Surgery	Wound Infection	Organism cultured in wound
1	Bose	9191	64	M	+	-	-	-	-	CAL	E-COLI	EARLY	-	-
2	Devi	16709	37	F	+	+	-	-	+	CAL	STAPH	EARLY	+	STAPH
3	Sakthivel	20416	37	M	+	-	-	-	+	CAL	-	ELECTIVE	+	STAPH
4	Muthukumar	21623	35	M	+	-	+	-	-	ACAL	-	EARLY	-	-
5	Marimuthu	808	38		+	-	-	-	-	ACAL	-	ELECTIVE	-	-
6	Maideen	6457	48	M	+	+	-	-	-	CAL	-	EARLY	-	-
7	Mookan	8607	65	M	+	+	-	-	-	CAL	-	EARLY	-	-
8	John Bosco	7900	43	M	+	-	-	-	-	CAL	E.COLI	ELECTIVE	-	-
9	Chithra	8700	40	F	+	+	+	-	-	CAL	E.COLI	ELECTIVE	-	-
10	Maruthayee	7899	65	F	+	+	+	-	+	CAL	STAPH	EMERGENCY	-	-
11	Sivagami	8151	44	M	+	+	-	-	-	CAL	-	EARLY	-	-
12	Kumaresan	12791	43	M	+	-	-	-	-	CAL	E.COLI	ELECTIVE	-	-
13	Mumtaj	14294	40	F	+	+	+	-	-	CAL	E.COLI	ELECTIVE	-	-
14	Pichai	15567	45	M	+	+	+	-	+	CAL	-	EMERGENCY	-	-
15	Kanthamani	16221	55	M	+	+	+	-	+	CAL	STAPH	EMERGENCY	-	-

16	Ellammal	033171	60	F	+	-	-	-	-	CAL	E-COLI	EARLY	-	-
17	Nagammal	030575	42	F	+	+	-	-	+	CAL	STAPH	EARLY	+	STAPH
18	Kuppammal	019710	65	F	+	-	-	-	+	CAL	STAPH	ELECTIVE	+	STAPH
19	Sengammal	003259	40	F	+	-	+	-	-	ACAL	-	EARLY	-	-
20	Nalammal	007321	45	F	+	-	-	-	-	ACAL	-	ELECTIVE	-	-
21	Venkatiah	005635	51	M	+	+	-	-	-	CAL	-	EARLY	-	-
22	Devaraj	040424	48	M	+	+	-	-	-	CAL	-	EARLY	-	-
23	Pairabee	011523	46	F	+	-	-	-	-	CAL	E.COLI	ELECTIVE	-	-
24	Vijayalakshmi	001432	22	F	+	+	+	-	-	CAL	E.COLI	ELECTIVE	-	-
25	Rani	011338	34	F	+	+	+	-	+	CAL	STAPH	EMERGENCY	-	-
26	Primila	22101	23	F	+	+	+	-	+	CAL	-	EMERGENCY	-	-
27	Thomayammal	22016	60	F	+	+	+	-	+	CAL	-	EMERGENCY	-	-
28	Ramaraj	24018	48	M	+	+	+	-	+	CAL	-	EMERGENCY	-	-
29	Paramasivam	26208	35	M	+	+	+	-	+	CAL	STAPH	EMERGENCY	-	-
30	Menaga	27856	25	F	+	-	-	-	-	CAL	E-COLI	EARLY	-	-
31	Mariyappan	16387	60	F	+	+	-	-	+	CAL	-	EARLY	+	STAPH
32	Abitha	31455	27	F	+	-	-	-	+	CAL	-	ELECTIVE	+	STAPH

33	Lakshmi	34659	56	F	+	-	+	-	-	ACAL	-	EARLY	-	-
34	Moorthy	32228	37	M	+	-	-	-	-	ACAL	-	ELECTIVE	-	-
35	Elizhabath Rani	34017	36	F	+	+	-	-	-	CAL	-	EARLY	-	-
36	Subashini	34484	25	F	+	+	-	-	-	CAL	-	EARLY	-	-
37	Rani	36766	49	F	+	-	-	-	-	CAL	E.COLI	ELECTIVE	-	-
38	Selvam	37518	34	M	+	+	+	-	-	CAL	E.COLI	ELECTIVE	-	-
39	Karuppaiah	39639	60	M	+	+	+	-	+	CAL	STAPH	EMERGENCY	-	-
40	Balayayee	39057	45	F	+	+	+	-	+	CAL	STAPH	EMERGENCY	-	-
41	Muthu	40639	49	M	+	+	+	-	+	CAL	STAPH	EMERGENCY	-	-
42	Mahamayee	43032	50	F	+	-	-	-	-	ACAL	-	ELECTIVE	-	-
43	Reginamary	40732	48	F	+	+	-	-	-	CAL	-	EARLY	-	-
44	Jeyalakshmi	41326	45	F	+	+	-	-	-	CAL	-	EARLY	-	-
45	Thayammal	43571	45	F	+	-	-	-	-	CAL	E.COLI	ELECTIVE	-	-
46	Kumar	43732	76	M	+	+	-	-	-	CAL	-	EMERCENCY	-	-
47	Thiru	42326	23	M	+	+	+	+	-	ACAL	-	EMERCENCY	-	-
48	Shanmugam	38527	51	M	+	-	-	-	-	CAL	-	EARLY	-	-
49	Abdul	27506	58	M	+	-	-	-	+	CAL	-	EARLY	-	-
50	Balakrishnan	35927	30	M	+	+	-	-	-	CAL	-	EARLY	-	-